7 Gender and Psychophysiology of PTSD

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For many men and women, traumatic life events exact a dramatic and sometimes enduring toll on emotional functioning. As one example, even when encountered months or years after a traumatic event, life experiences that are reminiscent of the event may revivify traumatic emotion and generate increases in heart rate, muscle tension, and other autonomic functions. Indeed, elevated autonomic responses to trauma-reminiscent "cues" are a hallmark characteristic of posttraumatic stress disorder (PTSD) that develops in the wake of overwhelming life events such as combat or rape (e.g., Kardiner, 1941).

Historically, psychophysiological studies of autonomic responses played a critical role in the emerging science of PTSD, providing empirical validation for patients' clinical reports of heightened physical stress and tension (Blanchard & Buckley, 1999). Psychophysiology is likely to play an equally important role in shaping future scientific inquiry into some of the central questions about PTSD, including its clinical course, responsiveness to clinical interventions, and accompanying medical comorbidities. For instance, psychophysiological responses are recognized as potentially important prospective predictors of emotional sequelae and clinical status following traumatic exposure (e.g., Blanchard et al., 1996), and preliminary evidence suggests they may be valuable process indicators of psychological treatment efficacy (e.g., Pitman et al., 1996). In addition, exaggerated cardiovascular responses to psychological stressors are identified as potential contributors to physical morbidity and mortality, particularly cardiovascular diseases such as atherosclerosis and elevated blood pressure (e.g., Barnett, Spence,

Manuck, & Jennings, 1997). Thus, psychophysiology may be central to mapping the processes that contribute to the emerging links among traumatic stress exposure, PTSD, and compromised physical health status (e.g., Schnurr & Jankowski, 1999). Finally, and perhaps most fundamentally, psychophysiological responses are one element of a more broadly integrated construct of emotional functioning, making them key to any comprehensive understanding of the alterations in basic emotional dynamics that are so pervasive in PTSD, particularly in its chronic forms.

Within this context, the present chapter considers the scientific literature on gender and psychophysiology of PTSD among adults. Although there is a sizable empirical literature on the psychophysiological responses of men with PTSD, empirical study of the psychophysiology of PTSD among women is relatively new. Thus, one aim of this present chapter is to comprehensively review studies of the psychophysiology of adult women with PTSD, including the few studies that have included both men and women. In addition, drawing upon the stress and emotion literature more broadly, we consider a triad of biopsychosocial factors important for a comprehensive understanding of the gender and psychophysiology of PTSD. This biopsychosocial triad—reproductive hormones, emotions and coping, and chronic environmental strain—has implications for understanding the autonomic consequences of trauma-reminiscent experiences and the health consequences of exaggerated autonomic responding, and for investigations designed to illuminate gender differences in the psychophysiology of PTSD. Finally, drawing from this literature review, we make recommendations for the next generation of research on gender and the psychophysiology of PTSD.

PSYCHOPHYSIOLOGY OF PTSD: A BRIEF HISTORY AND OVERVIEW

Prior to formal recognition of PTSD as a diagnostic entity, the sequelae of combat trauma exposure were described as irritable heart, shell shock, or war neurosis. Many reports of these disorders referred to elevated autonomic arousal as part of the symptom profile. Dobbs and Wilson (1960) provided the seminal laboratory examination of this phenomenon when they examined heart rate (HR), respiration rate, and electroencephalograph (EEG) responses of World War II and Korean combat veterans, and a comparison group of university students, while participants were exposed to simulated combat sounds. The combat veterans had higher baseline levels of HR and respiration rate and also showed a greater magnitude of cardiac response to the combat sounds than the students.

Subsequent studies explored the relationship between PTSD diagnostic status and physiological reactivity in cue reactivity paradigms with a PTSD

group, a trauma-exposed non-PTSD comparison group, and occasionally, a non-trauma-exposed comparison group. Procedurally, participants in these studies were presented with a variety of trauma- and non-trauma-related stimuli while their peripheral physiological responses were recorded. Independent laboratories replicated the general finding that, relative to those in comparison groups, individuals diagnosed with PTSD showed elevated physiological responses to trauma-related stimuli. This was observed across multiple physiological indicators, including cardiovascular, electrodermal, and muscle tension reactivity (e.g., Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). As a whole, these early studies indicated that the elevated autonomic arousal associated with PTSD was fairly specific to trauma-related stimuli and did not generalize to all classes of stimuli (e.g., Pitman et al., 1987).

In the ensuing years, empirical studies across populations, laboratories, and experimental methods have consistently demonstrated that when compared to PTSD diagnoses obtained through the use of structured interviews, autonomic reactivity measures discriminate between cases and noncases with sensitivity and specificity greater than .70. Thus, researchers have argued that physiological responses to trauma-related cues may be a useful adjunct to standard assessment procedures (e.g., Blanchard & Buckley, 1999).

More recently, investigators pursuing an independent line of research have sought to validate the clinical observation that individuals diagnosed with PTSD demonstrate an enhanced *startle* response to external stimuli. The evidence for this phenomenon has been less persuasive than the cue reactivity research; some studies have found evidence for exaggerated startle responses (Orr, Lasko, Shalev, & Pitman, 1995), whereas others fail to find support for the phenomenon (Grillon, Morgan, Southwick, Davis, & Charney, 1996). Recent work has focused on how the startle response, which is primarily mediated by brainstem neural circuitry (LeDoux, 1990), is modulated by higher cortical influences, such as negative emotional states induced by the presence of explicit threat cues (Grillon, Morgan, Davis, & Southwick, 1998). It may be that elevated startle responses and autonomic reactivity to trauma-related cues are functionally related. For example, priming negative affective states with exposure to trauma-related cues may potentiate startle responses to neutral stimuli.

APPLICATIONS OF PSYCHOPHYSIOLOGY: CLINICAL STATUS, TREATMENT EFFICACY, AND CARDIOVASCULAR HEALTH

In addition to its role in assessment and symptom validation, psychophysiological measurement has the potential to yield important information in applied domains, including predicting the clinical course of PTSD

and indexing the efficacy of clinical interventions designed to ameliorate its symptoms. In addition, because exaggerated cardiovascular responses to psychological stressors have been identified as potential contributors to physical morbidity and mortality, psychophysiological studies of PTSD may be essential to understanding linkages among traumatic stress exposure, PTSD, and compromised physical health status (Schnurr & Jankowski, 1999).

Prediction of Clinical Status and Response to Treatment

Assessment of physiological responses may have utility in predicting at-risk individuals immediately after trauma exposure. For example, Blanchard et al. (1996) found that cardiac response to trauma-related cues in the months shortly after a motor vehicle accident (MVA) predicted future clinical PTSD status 12 months posttrauma. MVA survivors who showed the greatest cardiac reactivity to trauma cues shortly after a traumatic event were those with the highest probability of meeting diagnostic criteria for PTSD at a 12-month follow-up. Similarly, elevated resting HR assessed during the acute posttrauma period predicted PTSD at 1 month (Shalev et al., 1998) and 6 months after the trauma (Bryant, Harvey, Guthrie, & Moulds, 2000).

In addition to predicting follow-up clinical status, physiological response to trauma-related cues may also index treatment response. Many contemporary behavioral and cognitive-behavioral accounts of anxiety or PTSD assert that physiological arousal and diminution of response to trauma-related cues are key markers for monitoring the efficacy of intervention (e.g., Foa, Steketee, & Rothbaum, 1989). Accordingly, some investigators have examined whether physiological reactivity to relevant cues and reduction in such responses during the course of treatment are associated with positive clinical outcomes with PTSD patients. Although the data are limited, existing studies suggest that psychophysiological responses measured during the course of treatment may have predictive utility (Blanchard & Buckley, 1999). Preliminary data suggest that combined within- and between-session reductions in physiological responses to trauma-related stimuli may predict more favorable improvements in clinical status (Pitman et al., 1996).

Cardiovascular Health

PTSD has been associated with increased incidence of myocardial infarction and elevated frequency of atrioventricular conduction problems (Boscarino & Chang, 1999), along with lower HR variability (Cohen et al., 1997), a risk factor for increased risk for mortality from cardiovascular events (Odemuyiwa et al., 1991). The potential for psychophysiological as-

sessment to illuminate linkages between PTSD and cardiac disease, and perhaps other medical conditions, is highlighted by laboratory and epidemiological investigations outside the domain of traumatic stress studies.

For example, elevated resting HR and blood pressure have been linked with future cardiovascular morbidity and mortality (e.g., Greenland et al., 1999). Other prospective, longitudinal studies have documented associations between elevated cardiovascular reactivity to mental and behavioral stressors, and occurrence of future blood pressure elevations (Matthews, Woodall, & Allen, 1993), hypertension (Menkes et al., 1989), coronary heart disease (Keys, Taylor, Blackburn, Anderson, & Somonson, 1971), and progression of carotid artery disease (Barnett et al., 1997). Exaggerated reactivity may also play a role in poor health more generally, including increased occurrence of minor infectious illnesses among otherwise healthy young adults (Dembroski, MacDougall, Slatts, Eliot, & Buell, 1981). These findings do not conclusively establish that cardiovascular parameters play a causal role in disease (e.g., reactivity may be a biological diathesis that promotes disease only when accompanied by stress exposure, or a marker for other cardiovascular disease risks), but they nonetheless highlight the role of psychophysiological assessment in evaluating pathways leading from stress to health outcomes (e.g., Boyce et al., 1995).

Results of these studies with nontraumatized populations suggest that connections among PTSD, cardiovascular parameters, and relevant health outcomes should be explored. When compared to individuals in control groups, both men and women diagnosed with PTSD show elevated cardiovascular responses to trauma-reminiscent cues (Pitman et al., 1987), along with elevated ambulatory and resting cardiovascular levels, particularly HR and, to a lesser extent, blood pressure (e.g., Keane et al., 1998; Orr, Lasko, et al., 1998). Although other studies have not found statistically significant group mean differences (e.g., Orr, Meyerhoff, Edwards, & Pitman, 1998), a recent meta-analysis of 34 studies found that, relative to individuals without PTSD, individuals diagnosed with PTSD have elevated resting HR and diastolic blood pressure (DBP) levels (Buckley & Kaloupek, 2001).

Speculatively, recurrent adrenergic response to stressful environmental cues and chronic physiological hyperactivation among individuals with PTSD may contribute to cardiovascular disease risk, perhaps by generating functional and morphological changes in the cardiovascular system (e.g., Blascovich & Katkin, 1993). Researchers are also beginning to examine the effect of PTSD on health outcomes in a variety of organ systems besides the cardiovascular system (e.g., Schnurr, Spiro, & Paris, 2000). Psychophysiological investigations that assess the impact of PTSD on sympathetic nervous system and neuroendocrine responses to environmental demands will clearly have a role in ascertaining the functional mechanisms by which PTSD becomes associated with poor health outcomes.

PSYCHOPHYSIOLOGY OF PTSD IN WOMEN

The assessment of physiological responses to both trauma-related and neutral stimuli has enhanced understanding of PTSD symptom presentation and can be a meaningful component of the comprehensive assessment of PTSD (Keane, Weathers, & Foa, 2000). In addition, emerging areas of inquiry highlight the relevance of psychophysiological assessment for understanding the course of PTSD, its responsiveness to mental health interventions, and its physical health correlates. However, the vast majority of psychophysiological studies of PTSD have been conducted with adult male participants; psychophysiological studies of women with PTSD began to appear in the early 1990s (e.g., Resnick, Kilpatrick, & Lipovsky, 1991). To date, approximately eight studies have examined autonomic reactivity and acoustic startle response in adult women with PTSD. We review this literature with a focus on two questions: (1) Do women with PTSD demonstrate elevated psychophysiological responding when compared to women without PTSD? (2) Do women with PTSD demonstrate the same pattern and magnitude of psychophysiological response as men with PTSD?

Psychophysiological Responding in Women with and without PTSD

Overall, when exposed to trauma-related cues, women with current PTSD demonstrate heightened psychophysiological levels (i.e., absolute response levels during cue exposure) and reactivity (i.e., increase from resting baseline) compared to women with lifetime occurrence of PTSD (i.e., women who once met but no longer meet diagnostic criteria), or women who never developed PTSD. As detailed later, group differences are exhibited in response to both trauma cues and startle probes, and across a variety of psychophysiological channels, including HR, skin conductance (SC), blood pressure, and muscle tension. A few studies, however, have failed to replicate group differences in psychophysiological responding.

In a study of women exposed to war-zone stressors in Vietnam, HR, SC, and systolic (SBP) and diastolic blood pressure (DBP) were measured during presentation of neutral and standardized Vietnam war-zone cues (Wolfe et al., 2000). On average, women with current war-zone-related PTSD exhibited significantly greater SC levels and SBP reactivity to the war-related cues than women who had never developed PTSD. The psychophysiological responses of the lifetime PTSD group generally fell between those of the other two groups. In another study of women Vietnam veterans, Carson et al. (2000) limited their sample to nurses who witnessed severe injury or death. Women with current PTSD were compared to women with no history of PTSD on HR, SC, and lateral frontalis and corrugator electromyogram (EMG) responses to audiotaped scripts of person-

alized and standard war-zone nursing scenes, as well as scripts with positive and neutral content. The PTSD group demonstrated greater HR, SC, and lateral frontalis EMG reactivity to both the personalized and standard trauma scripts than the group with no history of PTSD. Furthermore, women with PTSD showed greater HR and corrugator EMG reactivity to the personalized scripts than women without PTSD, despite both groups reporting equivalent emotional responses to the scripts (e.g., sadness and disgust). In addition, using an a priori discriminant function derived from psychophysiological studies conducted primarily with male participants (e.g., Pitman et al., 1987), the authors successfully classified 76% of the women with PTSD as responders (sensitivity) and 81% of women without PTSD as nonresponders (specificity).

Examining the effects of PTSD related to childhood sexual abuse, Orr, Lasko, et al. (1998) recorded the HR, SC, and EMG of women with current PTSD, women with lifetime PTSD, and women with a history of childhood sexual abuse who had never developed PTSD. Women with current PTSD exhibited higher HR during individually tailored imagery of their sexual abuse than did women in the other groups; they also manifested higher corrugator EMG levels than the women who never developed PTSD. Utilizing the same discriminant function described earlier, the authors found moderate levels of sensitivity (66%) and high levels of specificity (78%).

In contrast to these studies, two cue reactivity investigations failed to find consistent patterns of psychophysiological responding to trauma-related stimuli in women. Peirce et al. (1996) examined HR, SBP and DBP, SC, skin temperature, and lateral frontalis EMG response to standardized sexual assault cues in women with lifetime sexual assault-related PTSD, and women with no history of sexual assault-related PTSD (although some had sexual assault histories). There were no group differences in reactivity or absolute levels of any psychophysiological variable, although the lifetime PTSD group reported stronger negative emotional responses (e.g., angry, unhappy) to the trauma cues than did the control group. Lifetime rather than current PTSD status, use of standardized as opposed to idiographic cues, and population characteristics (e.g., all women had taken methadone prior to the study session) may account for the absence of group differences. Kilpatrick, Best, Ruff, and Veronen (1984) studied rape victims' HR and SC responses to rape-related fear scenes (e.g., walking past men, etc.). They found little elevation in HR or SC arousal to these scripts, perhaps because the scripts were not sufficiently potent cues.

Finally, several studies have examined the acoustic startle reflex in women with PTSD, with mixed results. In a study comparing women with current sexual assault–related PTSD to women who were never assaulted and had no history of PTSD, women with PTSD exhibited greater eyeblink responses in the left eye than the right, whereas women in the comparison

group exhibited symmetrical eyeblink responses (Morgan, Grillon, Lubin, & Southwick, 1997). In addition, women with recent PTSD (from adult sexual assault) exhibited greater left eyeblink reflexes than those with long-standing PTSD (from childhood or adolescent sexual assault). The authors suggested that the effects of PTSD on startle reflex pathways may subside over time. Another interpretation is that early trauma may affect the development of startle reflexes differently than later trauma. In contrast, eyeblink responses to acoustic startle probes did not differentiate women with current childhood sexual abuse-related PTSD, women with lifetime PTSD, and women with childhood sexual abuse who never developed PTSD (Metzger et al., 1999). However, women with current or lifetime PTSD had greater HR reactivity and slower habituation of SC responses to startling tones than women who never developed PTSD (Metzger et al., 1999). Among former Vietnam War nurse veterans, eyeblink response, SC reactivity, and SC habituation rate in response to startling tones failed to differentiate women with war-zone-related PTSD, women with lifetime PTSD, and women who never developed PTSD (Carson et al., 1999). Women with current PTSD demonstrated greater HR reactivity to startling tones than did the other two groups.

Psychophysiological Responding in Men and Women with PTSD

Most investigators who have included both men and women in psychophysiological studies of PTSD have neither compared men's and women's responding nor reported results by gender (e.g., Blanchard et al., 1996). To our knowledge, only two studies of the psychophysiology of PTSD have compared results by gender.

In a study of Israeli men and women, HR, SC, and frontalis EMG reactivity to personalized scripts involving trauma-related imagery were assessed in individuals with current non-combat-related PTSD, and individuals with no history of PTSD (Shalev, Orr, & Pitman, 1993). Across gender, participants with PTSD showed greater HR and EMG reactivity to the trauma imagery than participants with no history of PTSD. Although gender comparisons were not statistically significant, women with PTSD exhibited 33% higher physiological responding than men with PTSD (differences on specific physiological channels were not reported). A second study also assessed Israeli civilians, with and without PTSD, who had experienced repeated Iraqi missile attacks during the Gulf War (Laor et al., 1999). Compared to individuals without PTSD, those with PTSD demonstrated greater HR, SBP, and DBP reactivity to a standardized, trauma-related script (i.e., a radio transmission broadcast during the original missile attack). Participants with PTSD did not respond differently from those without PTSD to positive (i.e., ocean waves), action (i.e., police car chase), neutral (i.e., party chatter) or fear (i.e., child screaming with dogs barking)

scripts. The authors found neither a main effect of gender nor an interaction of PTSD with gender on psychophysiological responding. Although limited to two studies, these findings suggest that men and women with PTSD respond similarly to trauma cues across psychophysiological systems.

Summary and Conclusions

Psychophysiological studies suggest that women with current PTSD do experience heightened responding compared to trauma-exposed women who never developed PTSD, as well as women without trauma histories. In some cases, women with current PTSD showed greater reactivity than did women with lifetime PTSD. The majority of these small studies support the presence of HR and SC reactivity, both in response to traumatic event cues (e.g., Wolfe et al., 2000) and to acoustic startle probes (e.g., Metzger et al., 1999). Preliminary evidence suggests that facial EMG may be another useful indicator of differences between women with and without PTSD (e.g., Orr, Lasko, et al., 1998).

Although two studies did not find PTSD group differences in HR and SC responding (e.g., Peirce et al., 1996), several possibilities might explain this discrepancy. Both studies were hampered by methodological limitations, including unique population characteristics and the use of cues that were standardized or otherwise not specific to PTSD events. Furthermore, these two disconfirming studies may mirror the literature on men, in which approximately 30–40% of men with PTSD are "nonresponders" who fail to exhibit psychophysiological reactivity to trauma-related cues (Keane et al., 1998). The relatively new research on the nonresponse phenomenon has examined the influence of participants' psychological traits, symptoms, and comorbid conditions, any of which could be operative in psychophysiological studies of women.

Acoustic startle studies have so far been inconsistent in their results, with some finding that women with PTSD demonstrate exaggerated eyeblink response to startle stimuli (Morgan et al., 1997) and others finding no group differences (e.g., Metzger et al., 1999). Previous research with men has produced similarly inconsistent results, although researchers have begun to suggest factors that may contribute to this inconsistency (Shalev, Peri, Orr, Bonne, & Pitman, 1997).

With regard to gender comparisons—whether men and women with PTSD exhibit similar patterns of psychophysiological responding—the two relevant studies reviewed found no statistically significant gender differences (Laor et al., 1999; Shalev et al., 1993). Furthermore, two other studies provide indirect support of gender similarities: Carson et al. (2000) and Orr, Lasko, et al. (1998) cross-validated their psychophysiological assessment in women using a discriminant function drawn from a male-dominant sample. In summary, preliminary evidence indicates that both men and

women exhibit similarly enhanced psychophysiological reactivity when presented with trauma-reminiscent cues, but relevant studies are few in number and additional research is needed to substantiate these findings.

GENDER AND THE BIOPSYCHOSOCIAL TRIAD: REPRODUCTIVE HORMONES, EMOTION AND COPING, AND CHRONIC ENVIRONMENTAL STRAIN

It is well established that psychophysiological responses are influenced by the combined effects of biological, psychological, and social factors (e.g., Jackson, Treiber, Turner, Davis, & Strong, 1999). Drawing on the stress and emotion literature more broadly, we consider a triad of gender-linked biopsychosocial factors that are important for a thorough understanding of gender and the psychophysiology of PTSD: (1) reproductive hormones, (2) emotions and coping, and (3) chronic environmental strain. This biopsychosocial triad has implications for the autonomic consequences of trauma-reminiscent experiences and the health consequences of exaggerated autonomic responding, and may also be important in investigations designed to illuminate potential gender differences in the psychophysiology of PTSD.

Hormonal Correlates of Women's Cardiovascular Stress Responses

Among healthy adults, there are both similarities and differences in men's and women's cardiovascular and neuroendocrine responses to acute laboratory stressors (Stoney, Davis, & Matthews, 1987). In general, men and women show similar DBP, urinary norepinephrine, and cortisol responses. In contrast, gender differences are apparent for HR, SBP, and urinary epinephrine; women show the higher resting HRs and tend to show greater HR reactivity, whereas men show the higher resting SBP, SBP reactivity, and urinary epinephrine. It has been hypothesized that differences in men's and women's levels of circulating reproductive hormones (i.e., estrogen, progesterone) may provide a plausible basis for these observed gender differences in cardiovascular reactivity; for example, there are estrogen receptors in the myocardium, and estrogen potentiates certain mechanisms of vasodilation (Gilligan, Quyyumi, & Cannon, 1994; Legato, 1997). This hypothesis has been evaluated by a number of studies that directly and indirectly examined the role of reproductive hormones in cardiovascular reactivity.

Menstrual Cycle

The normal female menstrual cycle, with its cyclic alterations in estrogen, progesterone, follicle-stimulating and luteinizing hormones, has been used

as a natural experiment to assess the role of hormones in women's cardiovascular reactivity. The most rigorous investigations verify menstrual cycle phase by using serum assays of multiple hormones, as opposed to less reliable approaches, such as basal body temperature, self-report, or estradiol assays only (Stoney, Owens, Matthews, Davis, & Caggiula, 1990).

In general, studies that have employed this stringent method find no significant menstrual cycle phase effects (luteal vs. follicular, or vs. menstrual, or both) for blood pressure or HR reactivity to laboratory stressors (e.g., Stoney et al., 1990), although there are exceptions (e.g., Miller & Sita, 1994). However, these null effects for menstrual cycle phase do not mean that reproductive hormones are unrelated to women's acute cardiovascular stress responses. When serum estradiol and progesterone were directly assayed, higher levels of circulating progesterone were associated with lower DBP reactivity, and higher levels of estradiol were associated with lower HR and SBP reactivity (Sita & Miller, 1996). At the same time, there were no main effects of menstrual cycle phase on cardiovascular responses. Thus, there are relationships between reproductive hormones and cardiovascular responses to stress, but these relationships do not appear to culminate in reliable menstrual cycle phase effects on reactivity. One possible exception to this concerns hemodynamic measures (e.g., cardiac output, total peripheral resistance). Some evidence suggests that these measures do vary by menstrual cycle phase (although the reasons for this are unclear), even when BP and HR are statistically equivalent across phases (e.g., Miller & Sita, 1994).

Natural, Surgical, and Experimentally Induced Menopause

Biologically, natural menopause refers to the cessation of menses and is characterized by various hormonal changes, including declines in estradiol levels. A number of studies have tested associations between naturally occurring menopausal status and psychophysiological reactivity to acute laboratory stressors among normal women. The most rigorous studies ascertain menopausal status via serum hormone assays. Two studies reported no association between menopausal status and either cardiovascular resting levels (Blumenthal et al., 1991; Saab, Matthews, Stoney, & McDonald, 1989), or reactivity to laboratory stressors (Blumenthal et al., 1991), whereas Stoney (1999) reported higher resting and task HR among premenopausal women as opposed to postmenopausal women. In contrast, other evidence suggests that postmenopausal women exhibit greater HR and blood pressure reactivity than premenopausal women, but effects are contingent on the laboratory stressor employed (e.g., Saab et al., 1989). In the one study that included men, there was evidence that postmenopausal women's SBP and DBP responses exceeded those of men (Owens, Stoney, & Matthews, 1993). This suggests that women's postmenopausal status may

reverse the well-documented gender patterns for SBP (i.e., that men's reactivity is greater than women's) and DBP reactivity (i.e., no gender differences; Stoney et al., 1987). In summary, there is conflicting evidence regarding associations between menopausal status and stress reactivity, but only a few studies have examined this issue. Inconsistencies could reflect a number of factors, including age differences between selected groups of pre- and postmenopausal women, the effects of early versus late menopause, or unobserved differences in underlying hormonal status.

In other approaches to assessing the role of women's reproductive hormones in cardiovascular responses, the effects of either surgically or pharmacologically induced menopause have been examined. In one study, women who underwent a hysterectomy with removal of the ovaries showed greater blood pressure reactivity than women who underwent a hysterectomy without removal of the ovaries (Stoney, Owens, Guzick, & Matthews, 1997). This provides indirect support for the role of reproductive hormones in cardiovascular reactivity, because surgical removal of the ovaries results in a greater decline in circulating ovarian hormones than does hysterectomy alone. Contrasting results were obtained in a study that examined effects of pharmacological suppression of ovarian hormones on stress reactivity of premenopausal women (Matthews, Berga, Owens, & Flory, 1998). Contrary to expectations, experimental suppression of ovarian hormone levels was not related to variations in cardiovascular responses to acute laboratory stressors.

Exogenous Estrogens: Oral Contraceptives and Hormone Replacement

Women's oral contraceptive use has been associated with altered resting cardiovascular parameters, including lower DBP and total peripheral resistance, and higher cardiac output, stroke volume, and SBP (Davis, 1999). Cardiovascular reactivity does not appear to be associated with oral contraceptive use among nonsmokers. However, the combined effects of being a smoker and using oral contraceptives are associated with significantly higher blood pressure reactivity (Davis, 1999). One study did fail to replicate the increase in cardiovascular reactivity among women who smoke and use oral contraceptives (Girdler, Jamner, Jarvik, Soles, & Shapiro, 1997). Interestingly, however, this study examined smokers and nonsmokers of both genders, and found that being a smoker was related to cardiovascular reactivity only among females; the cardiovascular responses of male smokers and nonsmokers did not differ. This is consistent with other reports that women's heart rate and blood pressure responses to smoking exceed those of men's (Stone, Dembroski, Costa, & MacDougall, 1990).

With regard to hormone replacement therapy (HRT) among postmenopausal women, some studies suggest that HRT is associated with

attenuated cardiovascular reactivity (e.g., Collins et al., 1982), whereas other studies report that HRT is associated with greater reactivity (Burleson et al., 1998). Inconsistencies could be due to a variety of methodological factors, including duration of HRT use and differences in HRT administration (oral vs. transdermal patch) that affect delivery and metabolic pathways (Burleson et al., 1998).

Summary and Implications

Among healthy adults not selected for trauma exposure, there is preliminary evidence that certain reproductive hormones affect women's cardiovascular reactivity to acute stressors. Higher circulating levels of estrogen and progesterone have been associated with blunted reactivity. When hormonal levels are reduced due to menopause, women show cardiovascular hyperreactivity in some cases, and one commonly observed gender difference (i.e., SBP reactivity greater among men than women) reverses. With regard to exogenous hormones, oral contraceptive use increases acute reactivity among women who also smoke. The acute reactivity consequences of HRT are harder to evaluate, because the few studies that have been conducted vary greatly in important methodological parameters.

These studies raise a number of important substantive and methodological considerations for research on gender and the psychophysiology of traumatic exposure and PTSD. For example, among women with PTSD, cardiovascular reactivity to trauma-related stimuli may be magnified postmenopause, and with the use of certain exogenous hormones. To the extent that elevated reactivity plays a role in subsequent cardiac disease (e.g., Barnett et al., 1997), these life transitions and health behaviors could contribute to elevated risks for poorer health among women with traumatic stress exposures (e.g., Golding, 1994). This hypothesis should be explored empirically. In addition, smoking status may be particularly relevant to the cardiovascular regulation of women with PTSD, and even more so among women who use oral contraceptives. This topic deserves increased attention given the high prevalence of smoking among women with histories of victimization and PTSD (Acierno, Kilpatrick, Resnick, Saunders, & Best, 1996).

Another consideration of particular relevance for women with PTSD concerns the role of hormonal and menstrual cycle irregularities in cardiovascular dysregulation. In the studies cited earlier, potential participants with hormonal and self-reported menstrual cycle irregularities were carefully identified and excluded from participation. For example, Stoney et al. (1990) had participants evaluate their menstrual cycle patterns for 3–6 months prior to the laboratory session. One-fourth of the women initially eligible were eventually excluded due to menstrual cycle irregularities observed during this presession monitoring. Consequently, the generally null

effects of menstrual cycle phase and reactivity cannot safely be generalized to women with detectable menstrual irregularities, including alterations in both hormonal level and time course. This is particularly relevant to women with histories of trauma exposure given the documented associations between trauma exposure and menstrual irregularities; community surveys indicate that these problems are up to twice as prevalent among women who have been sexually victimized (Golding, 1996). Moreover, provocative preliminary data suggest that women's premenstrual syndromes, including premenstrual dysphoric disorders, are associated with cardiovascular dysregulation characterized by a mixed pattern of hyper- and hyporeactivity compared to controls (e.g., Girdler et al., 1998). Notably, one such study showed a significant positive association between premenstrual dysphoric disorder and history of sexual abuse. Taken together, these studies highlight the importance of examining links between cardiovascular dysregulation and traumatic stress exposure, including the potential mediating role of altered reproductive hormone profiles.

Finally, there are important methodological implications related to the behavioral and hormonal factors reviewed earlier (i.e., menstrual irregularities and menstrual syndromes, menopausal status, smoking status, and oral contraceptive use). If not assessed or controlled in psychophysiological studies of PTSD and gender, these factors represent potential threats to internal validity. Observed cardiovascular differences between groups could be due to differences in menstrual dysfunction, or menopausal or smoking status, for example, rather than to gender, diagnostic, or stress exposure status. Future studies should assess both psychiatric and control groups on these factors, or, where relevant and feasible, consider matching groups.

Gender Differences in Emotion and Coping

Gender differences in emotional experience, expression, and coping are well documented and may contribute to differences in how men and women respond to events that revivify traumatic affect and how they manage the emotional aftermath of such events. In this section, we consider the potential role of gender-linked affective and cognitive processes in the psychophysiology of PTSD.

Emotional Experience and Expression

Compared to men, women are more likely to express emotion (Kring & Gordon, 1998) and to report a greater intensity of feeling (Brody & Hall, 1993). Specifically, women report more negative, inwardly directed emotions than do men (e.g., shame, guilt, sadness, fear, and anxiety; Kring & Gordon, 1998). In addition, although reports of absolute levels of happiness are equivalent across gender, women report less happiness than men when happiness is considered as a proportion of total emotional experience

(Mirowsky & Ross, 1995). These gender differences in emotional experience and expression are thought to be due to socialization factors rather than to innate biological differences (Manstead, 1992).

On the basis of overall gender differences in emotion, one might expect women, as compared to men, to exhibit stronger verbal, facial, and psychophysiologic responsivity to negative emotion cues. To the contrary, studies of the psychophysiology of emotion reveal that although women's verbal and facial emotion expressions tend to be at least equal to men's responses, men tend to be more physiologically reactive than women (Kring & Gordon, 1998; Lai & Linden, 1992). Furthermore, triggers for physiological reactivity tend to be differentially salient for men and women; women respond more to affective valence, and men respond more to arousal (Lang, Greenwald, Bradley, & Hamm, 1993). With regard to specific emotions, women exhibit greater psychophysiological reactivity to sadness and disgust, whereas men exhibit greater reactivity to anger and fear (Kring & Gordon, 1998). Furthermore, after experiencing intense emotion, men's physiological recovery is faster if they express the emotion, whereas women's recovery is more dependent on their use of a preferred method of managing strong emotion (Lai & Linden, 1992). Many of these gender differences may be due to women being particularly attuned and responsive to emotional aspects of interpersonal relationships.

Although gender differences in emotion research suggest that, overall, women may be less psychophysiologically reactive, the majority of research has been conducted using anger as the target emotion. In these studies, women tend to exhibit less reactivity, or patterns that differ from men (Earle, Linden, & Weinberg, 1999). Women may be more, or differentially, reactive to cues of interpersonal traumatic events that arouse emotions such as sadness or disgust. This scenario is consistent with results of Carson et al. (2000), wherein nurse veterans asked to imagine their Vietnam experiences were both psychophysiologically reactive and reported intense sadness. Women are more likely than men to be exposed to interpersonal traumatic events, such as sexual assault, which typically precipitate high levels of inwardly directed negative emotion (i.e., sadness and shame). Thus, reminders of sexual assault and other interpersonal traumas—whether in the laboratory or in everyday experience—may predispose women to exhibit stronger psychophysiological responses than men. Future research should explore the possibility that psychophysiological reactivity in men and women with PTSD will be amplified in the context of gender-salient cues and emotions.

Coping

Although the relationship between coping and psychophysiological responding is not yet clear, evidence suggests that each influences the other. An avoidant coping style, characterized by denial and repression of

thoughts or emotions, is generally understood to be less effective in reducing distress. In studies conducted primarily with men, adherence to an avoidant coping style predicts increased SBP reactivity to stressful tasks in the laboratory (Morrison, Bellack, & Manuck, 1985) and delayed recovery of normal HR after stressful tasks (Vitaliano, Russo, Paulsen, & Bailey, 1995). Women who report that they often use an avoidant coping style also exhibit greater blood pressure reactivity to laboratory tasks than those who use avoidance less often (Fontana & McLaughlin, 1998). Furthermore, suppression of strong emotion blunts cardiovascular responding during a stressor but increases the likelihood of stronger responding to a later stressor (Larkin, Semenchuk, Frazer, Suchday, & Taylor, 1998). Psychophysiological suppression research mirrors research on thought suppression, which has demonstrated that women with rape-related PTSD experience a rebound in rape-related thoughts after suppressing them for a short time (Shipherd & Beck, 1999). Future research on coping and psychophysiological responding should address this issue by including extended recovery periods to evaluate the effect of rebound thoughts and emotions.

However, gender differences do exist in certain aspects of coping and psychophysiological responding. Although men who suppress their anger demonstrate greater blood pressure reactivity to stressful laboratory tasks, there are no differences in reactivity between women who do and do not suppress anger (Vögele, Jarvis, & Cheeseman, 1997), suggesting that the effects of some coping strategies may be less applicable to women than others. The meaning of the stressor itself may also play a role, in that differences in reactivity may not be detectable when tasks presented in the laboratory are less relevant to women (Lash, Gillespie, Eisler, & Southard, 1991). Thus, although coping has not been fully evaluated in studies of psychophysiological responding in PTSD, use of avoidance to cope with a personally relevant trauma cue might alter the pattern of psychophysiological reactivity. Because women are generally more likely to use emotion-focused coping, including avoidance (Ptacek, Smith, & Dodge, 1994), the alterations may be more evident in women.

It should also be noted that in cue reactivity paradigms employed in the study of PTSD, lab stressors that involve exposure to trauma-related cues typically require passive coping (e.g., listening to a trauma-reminiscent script; Carson et al., 1999). In contrast, "control" stressors include both passive coping (e.g., listening to an emotionally neutral script; Carson et al., 1999) and active coping tasks (e.g., mental arithmetic; Orr, Meyerhoff, et al., 1998). In general, active and passive coping tasks generate qualitatively different physiological loads, with the former eliciting elevated SBP and HR (a presumed ß-adrenergic load), and the latter eliciting increases in DBP (a presumed α-adrenergic load; e.g., Saab & Schneiderman, 1993). On the whole, however, trauma-cued physiological reactivity has been eval-

uated only in tasks producing an α-adrenergic load, on which men and women typically demonstrate equivalent reactivity, leaving an entire dimension of physiological responding unevaluated. This is clearly an area for future research.

Gender differences in coping and psychophysiological responding may be even more complex, involving hormonal status or mood, or both. One study reported a relationship between coping style and blood pressure reactivity in female participants that was evident only during the premenstrual phase, suggesting a complex influence of hormonal status on coping and psychophysiological reactivity (Fontana & McLaughlin, 1998). Alternatively, coping style may not directly affect psychophysiological responding; the relationship may instead be mediated by mood and one's response to his or her mood. Preliminary work demonstrates that dysphoric women are more likely than nondysphoric women to use both avoidance coping and rumination in response to depressed mood (Sigmon, Hotovy, & Trask, 1996). Although coping is not independently correlated to SC levels after exposure to a negative social scene, rumination is positively correlated, suggesting the possibility of a mediational effect (Sigmon et al., 1996). Women report more rumination in response to depressed mood than men (Nolen-Hoeksema, Parker, & Larson, 1994), and so may exhibit greater reactivity when in that mood state.

Psychophysiological changes may themselves trigger coping responses. Studies drawn from the larger anxiety literature reveal that individuals readily identify and process interoceptive cues. Accurate feedback about (nontraumatic) fear-induced physiological responding increases fear habituation and produces lower absolute levels of fear (Telch, Valentiner, Ilai, Petruzzi, & Hehmsoth, 2000). Interestingly, individuals with anxiety disorders are more accurate than nondisordered individuals in their assessment of interoceptive cues, and acute anxiety is positively correlated with accuracy (Zoellner & Craske, 1999). Although coping strategies are not typically assessed in interoception studies, we speculate that participants process their own physiological information and cope with it in such a way that both their reactivity and fear decrease. Indeed, the process may be circular, wherein an attempt to cope with emotions focuses attention on physiological cues and improves accuracy, which then engages attendant coping strategies and reduces physiological responding. Future research should attempt to extend these findings to PTSD populations, because the results have important implications for the treatment of hyperarousal in PTSD.

Although we know little about gender differences in coping with PTSD, studies have found that male veterans with PTSD use more emotion-focused coping strategies (especially avoidance) in general and in response to a trauma reminder than veterans without PTSD (e.g., Fairbank, Hansen, & Fitterling, 1991). In general, greater use of emotion-focused coping in re-

sponse to trauma is associated with greater distress in both men and women (e.g., Valentiner, Foa, Riggs, & Gershuny, 1996).

In summary, avoidant coping is associated with reactivity to stressful tasks in men and women not selected for trauma exposure or PTSD. The relationship may be further mediated by type of task, hormonal status, or mood. Psychophysiological responding may also engage coping systems, although the potential for gender differences is unknown in this field. The predominant use of emotion-focused strategies in women with PTSD places them at greater risk of continued distress when compared to men, and may affect their psychophysiological reactivity differentially.

Chronic Environmental Strain

Chronic environmental strain refers to ongoing "background" stressors that tax one's coping abilities and resources. The sources of chronic strain are myriad; examples include ongoing interpersonal conflict, excessive workplace demands, poverty, unstable or unsafe housing, and exposure to racist, sexist, or other environments that are hostile to aspects of one's core identity.

With regard to psychophysiology, some evidence suggests that chronic environmental strain significantly potentiates physiological reactivity to acute stressors (Matthews, Gump, Block, & Allen, 1997). Importantly, exposure to traumatic stressors appears to contribute to some sources of chronic strain; trauma survivors may experience increased occupational instability, more frequent periods of unemployment, and reduced incomes (Fairbank, Ebert, & Zarkin, 1999). For some individuals, chronic strain may be an equal or larger contributor than trauma to later distress or PTSD (e.g., Sundquist, Bayard-Burfield, Johansson, & Johansson, 2000). To date, however, no studies have examined how chronic background strain might contribute to the psychophysiological reactivity of individuals with PTSD.

The inclusion of measures of chronic strain may be particularly important for psychophysiological studies of women with PTSD; recent studies reveal that, compared to men, women experience more chronic strain and are exposed to more minor and major (nontraumatic) life stressors, including workplace and interpersonal stressors (Davis, Matthews, & Twamley, 1999). Chronic strain associated with being a woman in the military increases risky health behavior such as illicit drug use and smoking (Bray, Fairbank, & Marsden, 1999). Several authors have concluded that women experience greater chronic stress related to their gender role socialization than men, which, in turn, precipitates greater overall psychological distress and physical health impairment (Watkins & Whaley, 2000).

Chronic strain is also relevant to psychophysiology of minority group members. Research with healthy individuals in the United States documents the presence of ethnic differences and interactions between

ethnicity and gender on psychophysiological responsivity. For example, African Americans manifest greater overall cardiovascular reactivity to laboratory stressors compared to Caucasians (Anderson, McNeilly, & Myers, 1992). Additionally, cardiovascular reactivity in African Americans is mediated by peripheral resistance factors, whereas reactivity in Caucasians stems from changes in cardiac factors (Anderson et al., 1992; Light, Turner, Hinderliter, & Sherwood, 1993). Chronic stress exposure in the lives of African Americans due to the consequences of social inequality (e.g., higher unemployment and poverty rates) is thought to cause subtle physiological impairment (Anderson et al., 1992). Although the mechanism is not yet known, chronic strain may increase the magnitude of cardiovascular reactivity or lower the threshold for a cue to provoke a physiological response.

Notably, ethnic differences previously documented in men have not always generalized to women (e.g., Light et al., 1993). For example, using a public speaking stressor, Saab et al. (1997) found that African American men were less reactive on measures of HR, SBP, and cardiac output than were African American women and Caucasian men and women. Due to a similar interaction on measures of psychosocial functioning (e.g., coping, hostility), the authors concluded that the differences probably stem from environmental and social rather than biological factors. Because some research groups have found no interaction between ethnicity and gender on cardiovascular reactivity (Jackson et al., 1999), however, the question still remains to be fully addressed.

Chronic strain could conceivably exacerbate minority group members' physiological reactivity to trauma-related experiences. Although this specific hypothesis has not been tested, two psychophysiological studies of Cambodian refugees may shed light on the pertinent issues, in spite of mixed results. In the first study, refugees with PTSD demonstrated greater HR increases to both trauma and nontrauma stimuli than did American Vietnam veterans with PTSD, Cambodians without current PTSD, veterans without current PTSD, or Americans with no history of trauma or PTSD symptoms (Kinzie et al., 1998). Although analyses were not conducted by gender, the authors reported that the strongest subjective emotional reactions were evident in Cambodian women both immediately following and a week after the experimental session. In contrast, one startle reflex study of young adult Cambodian American male and females found that, overall, Cambodians were less physiologically reactive on SC and eyeblink measures than Caucasians (Wright, Masten, Northwood, & Hubbard, 1997). In addition, although male and female Caucasians were similar in reactivity, male and female Cambodians differed significantly in eyeblink and SC reactivity. Although chronic environmental strain may be influential in studies of gender and the psychophysiology PTSD, research in this area is still developing and, as yet, inconclusive. Clearly, the impact of trauma-

related loss of resources, as well as race- and gender-related strain, should continue to be evaluated.

SUMMARY AND DIRECTIONS FOR FUTURE RESEARCH

Overall, the research on gender and psychophysiology of PTSD is lacking, as is a coherent conceptual framework for the existing research. In this chapter, we not only reviewed the current literature but also outlined a triad of biopsychosocial factors that may serve as a guide for future research in this area.

Given the paucity of research on gender and the psychophysiology of PTSD, the most pressing need is for the inclusion of women in psychophysiological studies of PTSD. If the research reviewed here is an indication, there may be few or no gender differences in cardiovascular and SC reactivity to trauma-related stimuli. However, the support for this conclusion is not yet substantial. One obvious difficulty with conducting gender comparisons in trauma and PTSD research is that men and women are likely to have PTSD in response to different types of trauma (i.e., sexual assault–related PTSD is more prevalent among women, whereas combat-related PTSD is more prevalent in men), as well experience trauma and PTSD at different ages (e.g., sexual assault is likely to occur at an earlier age than war-zone exposure). When trauma type is confounded with gender, it is very difficult to determine the basis of any gender-related differences. To address this issue, researchers should focus on PTSD from traumas experienced with equivalent frequency, and at equivalent ages, in both genders (e.g., natural or perpetrated disaster). After substantial research has documented the presence or absence of a gender difference in psychophysiological reactivity, further exploration is necessary to determine whether mechanisms by which reactivity is expressed differ by gender.

Even if the results of future investigations support the conclusion that there are no gender differences in reactivity to trauma cues in men and women with PTSD, the end result may be influenced by factors that exhibit gender differences, such as reproductive hormones, emotions and coping, and chronic environmental strain. The associations noted between levels of estrogen and progesterone, and cardiovascular reactivity suggest that hormonal factors should be studied in women with PTSD. Women with current PTSD have high rates of some of the risk factors for reproductive dysfunction, such as menstrual cycle irregularity, natural or induced menopause, smoking, and oral contraceptive use (Acierno et al., 1996; Golding, 1996). These factors are known to alter cardiovascular reactivity, but their influence on reactivity in women with PTSD is unknown. Future psychophysiological studies of PTSD might examine the influence of these risk factors by matching groups on these variables. When matching is not feasible

(e.g., because hormonal or health behavior factors are strongly correlated with trauma exposure and PTSD), efforts to measure these related factors and assess their contribution to physiological responding are encouraged.

Emotional and coping factors need to be explored as potential sources of gender differences in the psychophysiology of PTSD. There are reliable gender differences in the expression and psychophysiological correlates of emotion, and because different trauma types and characteristics are likely to elicit different emotions, distinct patterns of psychophysiological arousal may emerge for men and women. Studies could be conducted in which subjective experience of emotion, and facial expressions of emotion, are coupled with psychophysiological assessment in men and women with PTSD and trauma-exposed individuals without PTSD. We also recommend that future research in the psychophysiology of PTSD build on basic interpersonal psychophysiology paradigms (e.g., Newton, Bane, Flores, & Greenfield, 1999) to examine interpersonal processes that may trigger reactivity among individuals with histories of interpersonal violence. These processes may have particular relevance to gender-related outcomes, because women are particularly reactive to and affected by negative characteristics of the social environment, perhaps in part due to gender role socialization.

Coping and psychophysiological reactivity processes may well influence each other in healthy adults, but little is known about their relationship in men and women with PTSD. Examining the association between dominant coping styles and reactivity, and the influence of interoception may prove to be fruitful. Future studies would also benefit from the use of a variety of active and passive coping tasks, as well as measures of the effect of hormonal status and mood on psychophysiological reactivity to trauma-related cues.

Finally, the influence of chronic environmental strain on psychophysiological reactivity in PTSD is, as yet, unstudied. Measurement of chronic strain has implications for psychophysiological responses, especially for women and ethnic minorities, but poses conceptual and methodological challenges similar to those posed by gender comparisons per se; that is, trauma type, severity, and age at exposure may be confounded with ethnicity and gender, making unambiguous interpretations difficult, if not impossible. Nonetheless, pursuing areas such as responses to natural, accidental, or perpetrated disasters, as well as measuring the strain incurred by exposure to these traumas, may be one way to approach this topic. In addition, ethnic comparisons within gender may be feasible (e.g., assessing responses to sexual assault among women of different ethnicities, and responses to combat among men), and would make an important contribution to this understudied area.

Finally, it should be noted that interactions between all the biopsychosocial factors may prove influential in psychophysiological studies of PTSD. For example, individuals under chronic strain due to racism may

have to engage coping strategies more often, with fewer resources. The greater resulting physiological load may lead to exaggerated psychophysiological reactivity when they are exposed to stressful cues (Clark, Anderson, Clark, & Williams, 1999). Research that successfully measures and evaluates all three factors will ultimately be the most productive.

In summary, trauma takes a dramatic toll on the mental and physical well-being of men and women throughout the world. Continued study of the psychophysiology of trauma and PTSD will enhance understanding of adaptation to catastrophic stressors and support efforts to treat associated psychological and physiological sequelae. Inclusion of men and women and people of color in these psychophysiological studies will greatly increase the applicability of this research and enhance the scientific understanding of trauma and PTSD.

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